

# WEST Search History

DATE: Sunday, December 29, 2002

<u>Set Name</u>	<u>Query</u>	<u>Hit Count</u>	<u>Set Name</u>
		result set	
<b>side by side</b>			
	<i>DB=JPAB,EPAB,DWPI; PLUR=NO; OP=ADJ</i>		
L33	L31 and (reduc\$4 or oxidation or oxidiz\$3)	15	L33
L32	L31 and ((heat(w)shock) or hsp\$2)	1	L32
L31	L29 and (protein\$1 near2 (denatur\$4 or denaturation))	76	L31
L30	L29 and mitochondrial near uncoupl\$3	1	L30
L29	(cancer\$3 or tumor\$1 or tumour\$1 or malignan\$3 or neoplas\$3 or carcinoma\$1 or adenocarcinoma\$1)	85890	L29
	<i>DB=USPT; PLUR=NO; OP=ADJ</i>		
L28	L2 or l26	27	L28
L27	L25 and @prad<20000119	1	L27
L26	L25 and @ad<20000119	27	L26
L25	L14 and l8	30	L25
L24	L23 and l8	0	L24
L23	L14[ti,ab]	11867	L23
L22	L8[ti,ab]	0	L22
L21	L8 with l14	0	L21
L20	trinitrophenol same l14	5	L20
L19	L18 or l17	33	L19
L18	L16 and @prad<20000119	3	L18
L17	L16 and @ad<20000119	33	L17
L16	L15 with (treat\$4 or therap\$3 or therapeutic\$1)	36	L16
L15	L14 with l13	73	L15
L14	(cancer\$3 or tumor\$1 or tumour\$1 or malignan\$3 or neoplas\$3 or carcinoma\$1 or adenocarcinoma\$1)	74814	L14
L13	TNP or trinitrophenol	861	L13
L12	l8 same (TNP or trinitrophenol)	0	L12
L11	l8 with (TNP or trinitrophenol)	0	L11
L10	L8 same radiation	2	L10
L9	L8 with radiation	0	L9
L8	mitochondrial near uncoupl\$3	48	L8
L7	mitochondrial adj uncoupl\$3	33	L7
L6	L1 and hyperthermia	0	L6
	<i>DB=DWPI; PLUR=NO; OP=ADJ</i>		

L5	L3 and hyperthermia	1	L5
L4	L3 and hyperhtermia	0	L4
L3	Bachynsky[in]	9	L3
<i>DB=USPT; PLUR=NO; OP=ADJ</i>			
L2	L1 and hyperhtermia	0	L2
L1	Bachynsky[in]	9	L1

END OF SEARCH HISTORY

L30 ANSWER 28 OF 30 MEDLINE                          DUPLICATE 22  
ACCESSION NUMBER: 95016440 MEDLINE  
DOCUMENT NUMBER: 95016440 PubMed ID: 7931080  
TITLE: Apoptosis, but not necrosis, of infected monocytes is coupled with killing of intracellular bacillus Calmette-Guerin.  
AUTHOR: Molloy A; Laochumroonvorapong P; Kaplan G  
CORPORATE SOURCE: Department of Cellular Physiology and Immunology, Rockefeller University, New York 10021.  
CONTRACT NUMBER: AI-07012 (NIAID)  
                  AI-22616 (NIAID)  
SOURCE: JOURNAL OF EXPERIMENTAL MEDICINE, (1994 Oct 1)  
                  180 (4) 1499-509.  
                  Journal code: 2985109R. ISSN: 0022-1007.  
PUB. COUNTRY: United States  
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)  
LANGUAGE: English  
FILE SEGMENT: Priority Journals  
ENTRY MONTH: 199411  
ENTRY DATE: Entered STN: 19941222  
                  Last Updated on STN: 19970203  
                  Entered Medline: 19941102

AB We have examined the effect of killing of host monocytes infected with bacillus Calmette-Guerin (BCG) on the viability of the intracellular mycobacteria. Peripheral blood monocytes were infected in vitro with a single bacillus per cell and maintained in culture for 6-8 d to allow the bacilli to replicate. Replicating viable BCG were found singly in perinuclear vacuoles bounded by tightly apposed lipid bilayers. Monocytes were then exposed to toxic mediators that induced killing of cells as evaluated by  $^{51}\text{Cr}$  release into the culture medium. Both **hydrogen peroxide (H}\_2\text{O}\_2** (an inducer of cell **necrosis**) and adenosine triphosphate (ATP $4-$ ) (an inducer of cell apoptosis) treatment killed infected monocytes. H $_2\text{O}_2$ -induced killing had no effect

on BCG viability. ATP-induced cell death was accompanied by DNA fragmentation and nuclear condensation. Apoptosis was associated with a swelling of the phagocytic vacuoles which became multibacillary and with a reduction of BCG viability as enumerated by colony-forming units.

L30 ANSWER 24 OF 30 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.  
ACCESSION NUMBER: 1996:557935 BIOSIS  
DOCUMENT NUMBER: PREV199699280291  
TITLE: **Hydrogen peroxide induced liver cell necrosis** is dependent on AP-1 activation.  
AUTHOR(S): Xu, Y. (1); Bradham, C. A.; Brenner, D. A.; Czaja, M. J.  
CORPORATE SOURCE: (1) Marion Bessin Liver Res. Cent., Albert Einstein Coll.  
Med., Bronx, NY USA  
SOURCE: Hepatology, (1996) Vol. 24, No. 4 PART 2, pp. 236A.  
Meeting Info.: 47th Annual Meeting and Postgraduate  
Courses of the American Association for the Study of Liver  
Diseases Chicago, Illinois, USA November 8-12, 1996  
ISSN: 0270-9139.  
DOCUMENT TYPE: Conference  
LANGUAGE: English

L30 ANSWER 17 OF 30 MEDLINE                          DUPLICATE 13  
ACCESSION NUMBER: 1999015689 MEDLINE  
DOCUMENT NUMBER: 99015689 PubMed ID: 9801070  
TITLE: ATP converts necrosis to apoptosis in oxidant-injured endothelial cells.  
AUTHOR: Lelli J L Jr; Becks L L; Dabrowska M I; Hinshaw D B  
CORPORATE SOURCE: Department of Surgery, University of Michigan Medical School, Ann Arbor, USA.  
SOURCE: FREE RADICAL BIOLOGY AND MEDICINE, (1998 Oct) 25 (6) 694-702.  
Journal code: 8709159. ISSN: 0891-5849.  
PUB. COUNTRY: United States  
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)  
LANGUAGE: English  
FILE SEGMENT: Priority Journals  
ENTRY MONTH: 199901  
ENTRY DATE: Entered STN: 19990202  
Last Updated on STN: 19990202  
Entered Medline: 19990115

AB Cell death due to necrosis results in acute inflammation, while death by apoptosis generally does not. The effect of adenosine triphosphate (ATP) on the pattern of cell death induced by oxidants was examined in bovine endothelial cells. ATP levels were altered by hydrogen peroxide (H<sub>2</sub>O<sub>2</sub>), glutamine (Gln), and metabolic inhibition (MI), to determine if necrosis can be shifted to apoptosis during oxidant injury. The form of cell death was determined by fluorescence microscopic techniques and the pattern of DNA degradation on agarose gels. ATP levels were measured using the luciferase-luciferin assay. Apoptosis occurred with 100 microM H<sub>2</sub>O<sub>2</sub> without an alteration in ATP levels. ATP was significantly lowered with 5 mM H<sub>2</sub>O<sub>2</sub>, and **necrosis** occurred. MI, in combination with 100 microM H<sub>2</sub>O<sub>2</sub>, decreased ATP and resulted in **necrosis**. MI alone, however, did not cause cell death. Gln partially restored ATP levels in cells injured with 5 mM H<sub>2</sub>O<sub>2</sub> and resulted in a significant increase in apoptosis. DNA laddering on agarose gels confirmed the apoptotic changes seen by fluorescence microscopy. In summary, a threshold level of ATP 25% of basal levels is required for apoptosis to proceed after oxidant stress, otherwise necrosis occurs. Agents like glutamine that enhance ATP levels in oxidant-stressed cells may be potent means of shifting cell death during inflammation to the noninflammatory form of death--apoptosis.

L30 ANSWER 18 OF 30 MEDLINE                          DUPLICATE 14

L14 ANSWER 1 OF 4 CAPLUS COPYRIGHT 2002 ACS  
 ACCESSION NUMBER: 1998:621138 CAPLUS  
 DOCUMENT NUMBER: 129:240865  
 TITLE: Photodynamic therapy generated oxidative stress for temporal and selective expression of heterologous genes  
 INVENTOR(S): Gomer, Charles J.; Wong, Sam Keng Sum; Nehme, Angela Ferrario; Luna, Marian Coensgen  
 PATENT ASSIGNEE(S): Research Development Foundation, USA  
 SOURCE: PCT Int. Appl., 37 pp.  
 CODEN: PIXXD2  
 DOCUMENT TYPE: Patent  
 LANGUAGE: English  
 FAMILY ACC. NUM. COUNT: 1  
 PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9840105	A1	19980917	WO 1998-US4551	19980309 <--
W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, HU, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, TJ, TM, TR, TT, UA, UG, UZ, VN, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG				
AU 9864539	A1	19980929	AU 1998-64539	19980309 <--
AU 731502	B2	20010329		
ZA 9801948	A	19990909	ZA 1998-1948	19980309 <--
EP 1005375	A1	20000607	EP 1998-910249	19980309
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI				
JP 2001514658	T2	20010911	JP 1998-539661	19980309
PRIORITY APPLN. INFO.:			US 1997-815402 A	19970310
			WO 1998-US4551 W	19980309

AB The present invention describes a method for the selection and temporal expression of heterologous genes. The invention consists of methods to obtain selective and temporal expression of heterologous genes in target tissues. Promoters inducible by photodynamic therapy or heat are used to express genes of interest under conditions of heating or Photodynamic Therapy (PDT)-induced oxidative stress. Such promoters may comprise

those

from **heat shock protein** or glucose-regulated protein genes. Selective and temporal expression of heterologous genes (such as cytokines, toxins, tumor suppressor genes, antisense mols. and anti-angiogenic factors) are of significant therapeutic benefit in the treatment of tumors, vascular proliferation and tissue hypertrophy. Gene therapy targeted by **laser** induced heating, other heating sources (such as microwave, ultrasound or radiofrequency induced currents), or

PDT

enhances treatment effectiveness by inducing expression of therapeutic genes in a controlled and localized manner.

REFERENCE COUNT: 7 THERE ARE 7 CITED REFERENCES AVAILABLE FOR THIS